Biologic Effects of Lead in Domestic Animals

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ABSTRACT

Lead poisoning in cattle usually is the result of a single ingestion of a material containing a large quantity of lead. Poisoning in cattle also can result from the long-term ingestion of crops or pasture forage contaminated by lead settling out from fumes and dusts emitted from industrial lead operations. The latter is the principal source of poisoning for horses. Horses appear to be more susceptible than cattle to the long-term ingestion of lead. Whereas a daily intake of approximately 2 mg/kg can produce poisoning in horses, a daily intake of approximately 6-7 mk/kg is required to produce poisoning in cattle. Although the ingestion of small amounts of lead by food-producing animals may not result in clinical signs of lead poisoning, it should be emphasized that a small fraction of that ingested will be retained in the tissues and contribute to the dietary intake of man.

It generally is considered that lead is the most common cause of accidental poisoning in domestic animals. The condition is diagnosed most frequently in cattle and dogs. It should be kept in mind that a discussion of lead poisoning in domestic animals must differ from the approach taken for man. Whereas subtle, subclinical effects of lead are highly relevant and important for man, similar considerations in animals are not practical. Lead poisoning in animals usually is recognized only when overt clinical signs of poisoning are apparent. Nevertheless, it is emphasized that even though apparently non-toxic quantities of lead are ingested by food producing animals, some of the lead will be absorbed. This would result in the addition of a finite amount of lead to the dietary intake of man.

Sources of Lead

The natural curiosity and licking habits of cattle make any available lead-containing material a potential source of poisoning. Some of the sources incriminated include lead-base paint (either from discarded paint cans or paint peeling from walls), used motor oil, discarded oil filters, storage batteries, certain types of greases and putty, and linoleum (Hammond et al., 1956; Buck, 1970a). These sources have been incriminated on the basis of 1) evidence of ingestion, 2) clinical signs, and 3) finding elevated concentrations of lead in the tissues. These sources can be found in the vicinity of farm buildings and in dumps located in pastures. It is interesting that these sources rarely are incriminated in lead poisoning in horses. Horses are much more selective than cattle in their eating habits. They usually do not lick old paint cans, storage batteries, peeling paint, nor do they seem to find the taste of used motor oil attractive.

Common histories of exposure in dogs include chewing on objects painted with lead-base paints, (e.g., when home remodelling entails scraping of plaster and old paint), eating linoleum, or ingesting lead materials such as shotgun slugs or curtain weights (Zook et al., 1969). The latter objects are retained in the stomach where they are ionized to an absorbable form due to the

[1]For personal data, see footnote 1 to Dr. Aronson's paper entitled "Biologic Effects of Lead in Fish," this issue.–Ed.

acidity of the stomach. Dogs less than 6 months of age are affected more commonly than older dogs, but this may be related to the almost completely indiscriminate eating habits of younger dogs.

Several outbreaks of lead poisoning in domestic animals have been recorded in North America and throughout the world where the source of metal was contamination of pasture or crops by industrial lead operations (Haring and Meyer, 1915; Hughes, 1923; Miessner, 1931; Holm et at., 1953; Beijers, 1952; Hupka, 1955; Hammond and Aronson, 1964; Kradel et al., 1965; Harbourne et al., 1968). These outbreaks differ from the more common cases of lead poisoning described previously in that several animals may be involved. Pastures and crops are contaminated by fumes and dusts emitted from lead industries settling out on the surrounding countryside. Animals eating this vegetation can accumulate amounts of lead sufficient to produce clinical signs of lead poisoning. A number of studies have been made to determine if the lead found in vegetation is the result of direct airborne origin or due to translocation from soil. These studies recently have been reviewed by P. K. Mueller and R. L. Stanley (1970, pers. comm.). They conclude on the basis of their work and the work of others that translocation from soil does not contribute more than 15 μg/gm dry weight of forage even when plants are grown in soil containing up to 700-3000 μg/gm. Thus, amounts of lead in plants in excess of 15 μg/gm most likely are due to direct aerial fallout. The extent to which contamination can occur is illustrated by finding concentrations of 3200 μg/gm dry weight in corn leaves located 75 yards from a lead smelter in one outbreak (Hammond and Aronson, 1964).

Susceptibility to Lead

It has been possible to estimate that a daily intake of 6-7 mg/kg constitutes a minimum cumulative fatal dosage of lead for cattle (Hammond and Aronson, 1964). This intake represents a concentration approximately 300 ppm lead in the total diet. These cattle were located approximately 2 miles from the smelter, but were fed lead-contaminated hay and corn silage grown in fields adjacent to the smelter. A fatal case of lead poisoning occurred following approximately 2 months on this diet. An intake of approximately half this dosage had no observable effect on cattle at another farm the previous winter. In this connection it is of interest to note that daily dosages of 5-6 mg/kg have been fed to cattle for a period of 2 years with no observable clinical effects (Allcroft, 1950), but that longer intake at this rate may be fatal (Allcroft, 1951).

There is some evidence suggesting that horses may be more susceptible than cattle to the chronic ingestion of lead. Whereas horses contracted lead poisoning on pastures adjacent to a lead smelter in one outbreak, cattle grazing in the same area appeared healthy (Larsen, A.A., 1969, pers. comm.). At one farm adjacent to a smelter in another outbreak, horses succumbed to lead poisoning in March following a winter intake in their hay of 2.4 mg Pb/kg/day (Hammond and Aronson, 1964). It was not possible to determine lead intake from pasture grazing the previous summer. However, since cows and horses had similar pasture that summer, and since the winter ration for the horses contained appreciably less lead than did that for the cows, it would seem that cumulative toxicity occurred somewhat more readily in horses. It is of interest to consider that pasture grass containing in excess of 80 μg Pb/gm dry weight was toxic to horses in still another outbreak (Mueller and Stanley, 1970, pers. comm.). If one assumes the horses weighed 400 kg and ate 10 kg grass (dry weight) per day, a minimal toxic dosage could be estimated at 2 mg Pb/kg/day; a figure close to the previous estimate.

Although the evidence above does suggest that horses might be more susceptible to lead than cattle, a consideration of grazing habits of horses precludes any firm conclusions. Horses occasionally will pull forage out by the roots and eat the roots and attendent soil along with the forage. Cattle rarely, if ever, do this, probably because they lack the jaw structure which makes it possible. The soil near smelters usually con-

tains far greater amounts of lead than does the forage. It is apparent that a horse showing a marked tendency toward this habit could ingest far greater quantities of lead than would be estimated from the analysis of forage alone.

It is only natural that human beings residing close to smelters near which animals are dying of lead poisoning should be concerned about their own health. In many cases these people are eating produce from home gardens. It is noteworthy that analysis of blood and urine of these people by local public health officials has not revealed evidence of increased lead absorption. Keep in mind that horses and cattle are vegetarians. If their hay or pasture is contaminated with lead, their entire diet may consist of contaminated vegetation. Probably only a small fraction of the total diet of human beings would consist of food grown in the vicinity of a lead operation. Furthermore, it is customary for people to wash garden produce (or husk corn) before its consumption. This practice undoubtedly would remove appreciable quantities of surface lead deposits. Since the animal and human population near the smelters breathed the same air, and since residents in the area have not shown evidence of increased lead absorption, it may be justified to conclude that the animals received virtually all of their lead burden through oral ingestion.

Clinical Signs of Lead Poisoning

All domestic species with lead poisoning exhibit varying degrees of derangement of the central nervous system, gastrointestinal tract, muscular system, and hemopoetic system. Differences occur clinically, however, in the relative severity of signs referrable to these organs and tissues. The most striking syndrome is presented commonly by young calves. The calf may suddenly begin to bellow and stagger about with rolling eyes and frothing mouth and often blindly crashes into objects. This phase may last up to 2 hours before sudden collapse and death. With less severe cases, depression, anorexia and colic may be observed. The animals may be depressed, blind, grind their teeth, move in a circle, push against objects, and be ataxic. Adult cattle present the latter signs most frequently, although the syndrome of maniacal excitement is not uncommon.

The syndrome in sheep consists mainly of depression, anorexia, abdominal pain and usually diarrhea. Excitatory phases have never been reported for sheep. Anemia is common during chronic ingestion.

The syndrome in horses consists mainly of depression, stupor, knuckling at the fetlocks, and a laryngeal paralysis producing an obstruction in the air passage and causing the horse to "roar." Anemia is commonly associated with lead poisoning in horses (Clarke and Clarke, 1967).

Gastrointestinal and central nervous system signs are seen with almost equal frequency in dogs. At some time during the course of poisoning approximately 87% of dogs show gastrointestinal signs consisting of emesis, colic, diarrhea, and anorexia. Approximately 76% of dogs show central nervous system signs consisting of hysteria and convulsions. Anemia and basophillic stippling commonly are associated with lead poisoning in dogs and are considered to be of diagnostic significance (Dodd and Staples, 1956; Zook et al., 1969).

Abortions have been reported in ewes grazing lead-mining areas in England (Egan and O'Cuill, 1969). A high rate of abortions and failures to conceive were noted in ewes experimentally fed finely divided metallic lead at a rate sufficient to induce signs of intoxication (Buck, 1970b). The lethal dose of lead in pregnant ewes appears to be considerably lower than in non-pregnant ewes (Allcroft and Blaxter, 1950). Cattle and horses have given birth to normal offspring following excessive lead exposure (Shupe, 1967; Egan and O'Cuill, 1970), but the small number of animals reported (5) makes it impossible to state that lead has no effect on the fetus in these species.

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